

## COMMUNICATIONS

### THE DIAGNOSTIC VALUE OF BIOMICROSCOPY OF THE POSTERIOR PARTS OF THE EYE\*†

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I SHOULD like to thank you most cordially for the great honour you have done me in asking me to give the Montgomery lecture this year. Switzerland owes Ireland a deep debt of gratitude, because it was Irish monks who founded the first centres of learning in Switzerland. It is therefore an especially great honour for a Swiss to be allowed to come and lecture at your university.

I have chosen this subject because the biomicroscopy of the eye, a recent offspring of morphology, has contributed greatly during the last few decades to ophthalmological diagnosis, while more recently the biomicroscopy of the posterior segment has increased our understanding of some of the most important and most serious ocular diseases, and promises even more useful results in the future.

By biomicroscopy we understand, in ophthalmology, not only the microscopy of living tissues, but the method, developed by Gullstrand, of examining the transparent ocular media in the optical section of the focused intense beam of the slit lamp. This is a kind of ultra-microscopy with slight magnification. That we separate the biomicroscopy of the posterior parts of the eye, namely of the vitreous and the fundus, from that of the anterior parts is not an arbitrary distinction. The examination of the posterior parts of the eye requires, not only a slit lamp of high luminosity in which the axes of the microscope and the illuminating beam can form a very small angle, but also additional optical devices such as intermediate lenses or contact lenses. This is because the posterior parts of the eye can be observed with the microscope only when the refractive effect of the cornea is neutralized or when, by means of an additional convex lens, a real image of the posterior parts is formed, which can then be seen in the microscope. We ourselves use two kinds of contact lens, which offer great advantages with few drawbacks. The advantages are good magnification, an appreciation of depth that corresponds well with the actual conditions, a good reproduction of the natural curvature of the fundus, and accessibility to the outermost periphery of the retina. The chief disadvantage is that the lenses must be applied directly to the eye, a procedure not admissible in the case of recently

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operated patients and practically impossible to perform successfully with nervous or unruly children. In such cases we use the Hruby lens.

We use two contact lenses:

(1) To examine the central parts of the vitreous and the fundus we use a contact lens with a flat anterior surface with no scleral rim and a funnel-shaped holder (Fig. 1a).

(2) To examine the periphery of the fundus and vitreous we use a conical glass, containing three mirrors at various angles (Fig. 1b, c).

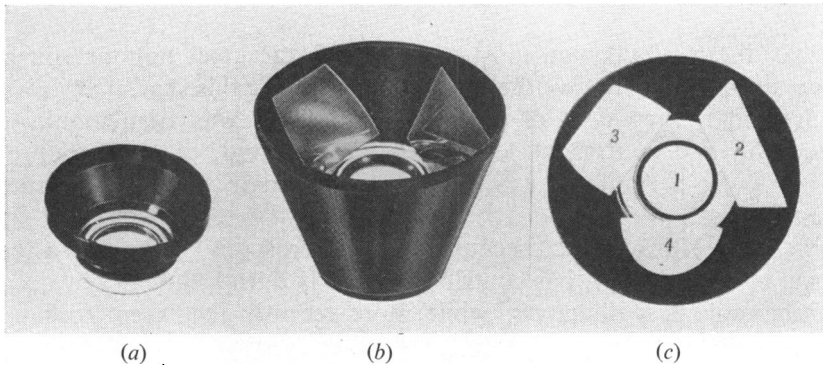


FIG. 1.—Contact lenses

(a) For examination of central part of fundus.

(b) and (c) Three-mirror glass seen from the side and from above.

There are four particular applications of biomicroscopy about which I propose to speak:

- (1) Retinal detachment.
- (2) Intra-ocular tumours.
- (3) Choroiditis.
- (4) Examination of the papilla in cases of special theoretical or practical interest.

(1) **RETINAL DETACHMENT.**—As an introduction to this subject I must discuss the normal vitreous, the structure of which can hardly be distinguished histologically in the adult. With the slit lamp the anterior vitreous membrane may be seen at the periphery, but axially it is attached to the lens. Behind this anterior margin of the vitreous may be seen a folded membrane, the so-called *membrana plicata*, which (as Mann has shown) is nothing but the limiting membrane of Cloquet's canal. It would be better to call this formation the primary vitreous. In the embryonic stage (Fig. 2, opposite) and in infancy it still forms a simple funnel which ends at the anterior vitreous membrane and runs straight backwards; in later life it hangs down and may be followed as an S-shaped structure through the whole vitreous as far as a point close to the papilla (Fig. 3, opposite). Especially clear is a narrow band which rises at about the middle of the vitreous and has led

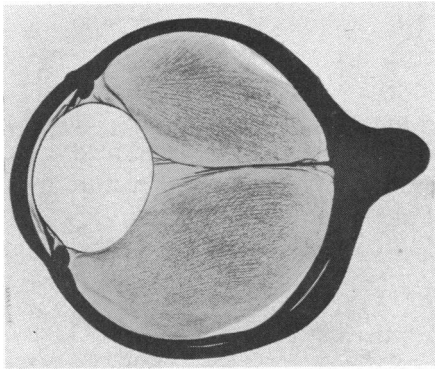


FIG. 2.—Vitreous with Cloquet's canal from an 18-mm. foetus (after *Jokl*).

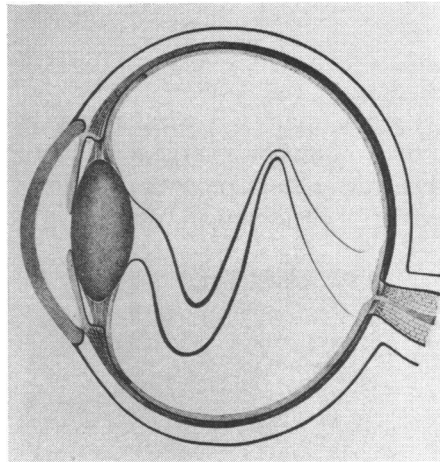


FIG. 3.—Diagram of course of Cloquet's canal in the adult.

some observers to think that there is a transverse membrane at the centre of this structure. In reality, however, this is only the narrow single or double band of the rising part of Cloquet's canal. In normal juvenile eyes, various sheets of the secondary vitreous—seen as bands in the cross-section—which scatter light to some extent are arranged around the primary vitreous (Fig. 4). At the embryonic stage there is no posterior vitreous membrane; this is formed after birth from submicroscopic elements lying one above the other.

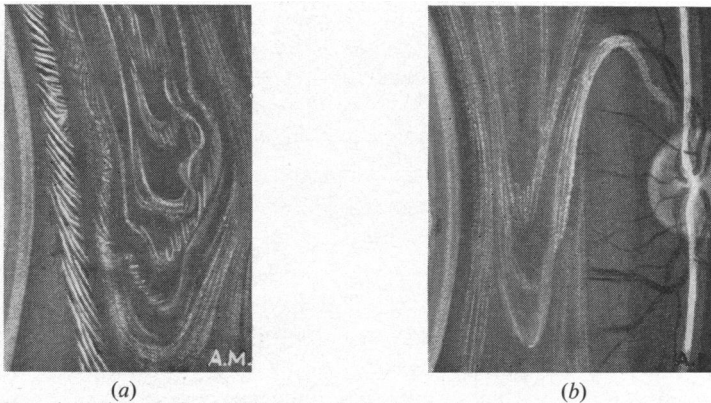


FIG. 4.—Vitreous structure around Cloquet's canal in a 7-year-old boy. (a) Without contact lens. (b) With contact lens.

In the course of life changes that are perceptible with the slit lamp occur in the jelly-like vitreous; in the above-mentioned bands, coarser fibres become visible and a process takes place which may be called fibrous destruction. Between the fibres cavities appear, and these are filled with a

viscous fluid, *i.e.* the jelly ceases to be of a homogeneous composition (Fig. 5). These cavities increase in size with age, and the fibres grow more numerous and coarser. The patient notices them as *muscae volitantes* (vitreous floaters). Flashes like lightning may be seen in the dark, and the day comes when a large *musca* is perceived in the temporal visual field. At this time a re-examination of the vitreous will give the typical picture of a posterior detachment of the vitreous (Fig. 6).

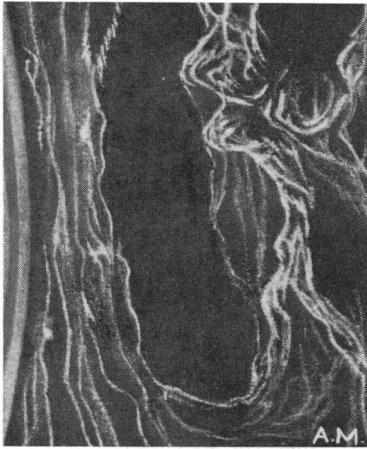


FIG. 5.—Typical vitreous destruction with cavity formation and appearance of coarse fibres.

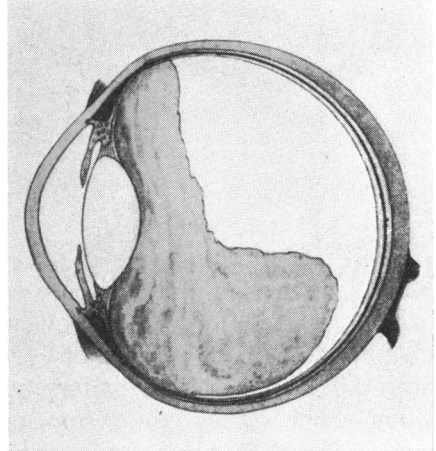


FIG. 6.—Diagram of vitreous detachment with collapse (according to Hruby).

Fig. 7 (opposite) shows how these vitreous changes develop with the passage of time. The vitreous detachment described (the vitreous detachment with collapse, Hruby), happens suddenly. In cases in which the vitreous had been examined beforehand, it was known that more or less large cavities had already existed before the sudden detachment, and afterwards these holes have almost completely disappeared, only a tangle of fibres remaining in the destroyed and collapsed vitreous. At the same time the fluid has left the vitreous and passed behind it. Sometimes one has the impression that the ring-shaped opacity that is so characteristic of vitreous detachment with collapse is also the opening through which the vitreous cavity has been emptied (Fig. 8, opposite) but I am not quite sure of this. However, it is certain that the process of vitreous detachment takes place rapidly, usually in a matter of minutes, or even of seconds, and rarely if ever in the course of an hour or more, and that considerable force is exerted in the process.

If the patient with a fresh vitreous detachment (24 to 48 hours old) comes to the ophthalmologist, in many cases fresh retinal tears can be seen, mostly in the outermost periphery of the retina, and even vitreous haemorrhages. This process of fibrous destruction, leading to cavity-formation and finally

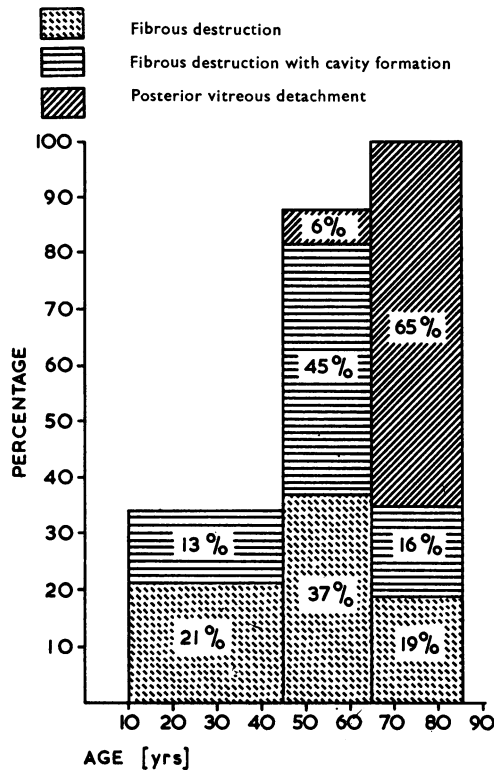


FIG. 7.—Age and vitreous changes.

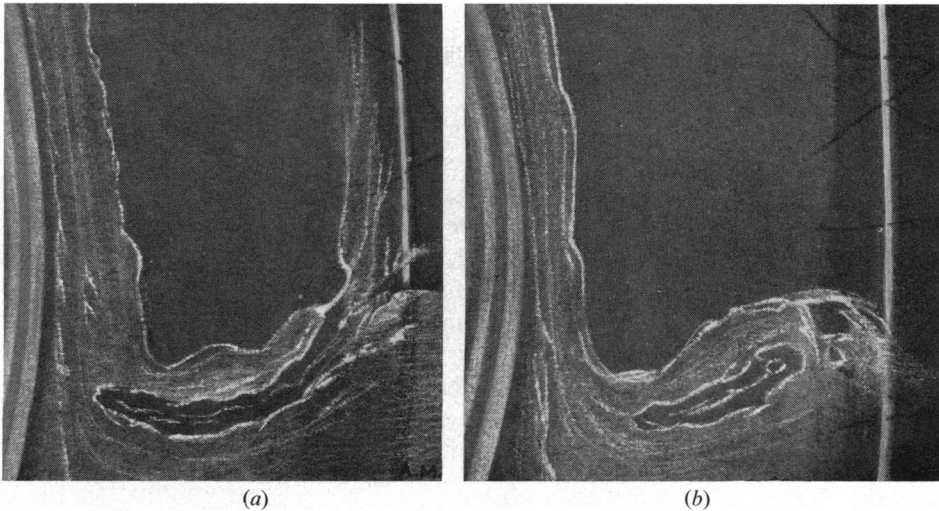


FIG. 8.—Vitreous detachment with collapse, showing communication between the collapsed vitreous cavity and the retro-vitreous space.

to vitreous detachment with collapse is a normal ageing process, so that only a few normal eyes over the age of 75 years have no vitreous detachment. It also occurs prematurely in patients with medium and severe degrees of myopia, on an average 20 years earlier than in emmetropes. Excessive cavity-formation has even been seen in 6-year-old myopes. On examining the periphery of the fundus after vitreous detachment, one sees the collapsed vitreous below resting on the fundus and the membrane above running in most cases towards the ora serrata.

In some cases, however, the vitreous adheres to the retina. These adhesions tend to occur at pathologically altered sites, at old choroiditic foci, in areas of cystic degeneration, and in areas where the peripheral vessels are obliterated (Gonin-Vogt lattice), but they also occur at sites that appear perfectly normal when magnified 10 to 20 times, especially in the region where the extra-ocular muscles (especially the superior obliques) are attached. The retinal tears that may be seen shortly after a vitreous detachment are nearly all "horseshoe" tears with flaps torn away from the wall, or else they are round holes with lids which float in the vitreous cavity. The flaps, like the lids, lie upon the detached posterior vitreous membrane (Fig. 9).

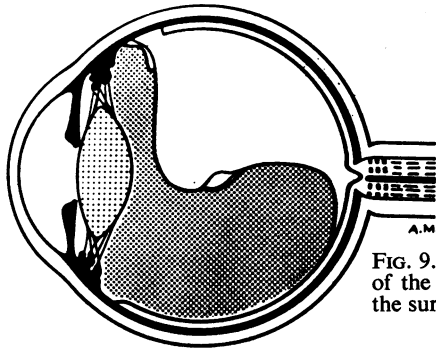


FIG. 9.—Diagram of horse-shoe tear of the retina. The flap adheres to the surface of the detached vitreous.

So long as no retinal detachment has followed, it is impossible to decide whether the hole is superficial or whether it pierces the entire thickness of the retina. If the periphery of the fundus is examined with the three-mirror glass after each fresh vitreous detachment, an astonishing number of tears can be discovered. On the other hand, such tears do not often occur later at the sites of such adhesions, *i.e.* months or years after the vitreous detachment. I have observed only one example of this second type compared with many cases of the first type. This led me to the conclusion that retinal tears of the "horseshoe" type or the lid type leading to retinal detachment nearly always occur at the moment when the vitreous becomes detached. Days, weeks, and even months may pass before a tear results in a retinal detachment which gives rise to clinical symptoms. This raises the question whether it is possible to foretell where a tear will appear. Since the vitreous is certainly attached at sites of cystic degeneration and in

Gonin-Vogt lattice areas, these sites are endangered by a vitreous detachment. But these affected sites are not the only ones where tears may occur. We have seen cases without vitreous detachment but with suspect retinal areas; prophylactic coagulations of all these sites were performed. When vitreous detachment occurred afterwards, retinal tears appeared at seemingly normal sites and were followed by retinal detachment.

The following case offers a most remarkable example:

A woman had a total retinal detachment in the right eye, new holes constantly being formed which could not be closed in spite of repeated operations. A careful examination of the second eye showed much fibrous destruction and cavity-formation in the vitreous but no vitreous detachment. Above, there was a Gonin-Vogt lattice formation, but otherwise the periphery was completely normal. It was proposed to wait a few weeks and then to coagulate that spot, but in the meantime the patient reported that typical signs of a vitreous detachment had appeared in that eye. Examination with the three-mirror glass showed a collapsed vitreous detachment, and at the periphery there were seven retinal tears, of which only two small ones were at the site of the degeneration, the others being at places formerly regarded as above suspicion. At one of these sites a small retinal detachment had already begun, but after immediate coagulation of all the holes, the eye remained normal.

It thus appears that peripheral retinal cysts, sites of cystic degeneration, and Gonin-Vogt lattices are places where retinal holes may occur in cases of vitreous detachment. But to coagulate these sites only will not necessarily prevent the development of a retinal detachment. If a vitreous detachment occurs afterwards, retinal holes may appear at quite unsuspected places.

It is therefore most important, for the prophylaxis of retinal detachment in patients who come to an ophthalmologist on account of fresh vitreous detachment, to examine the periphery of the retina with the three-mirror glass, and to coagulate all retinal tears which can be discovered. If vitreous detachment has already occurred, the degenerate regions of the retina to which vitreous adheres may be coagulated.

Besides the tears here described, which originate in connexion with vitreous detachment, there exist—less frequently—retinal holes which are unrelated to vitreous adhesions. These are due to retinoschisis and its consequences, a process which is still rather obscure. To this group also belong certain incomplete layer-holes, which are not dangerous at all—the so-called “macular” holes and Weber’s holes. In a series of 44 cases not one of these so-called “macular” holes was followed by retinal detachment. In some cases a retinal detachment had been caused by a real tear in the macula, but there was nothing in the history to suggest the previous existence of a “macular” hole. These macular layer-holes frequently result from cystic oedema. With the slit lamp it is easy to differentiate between hole and cyst: with a cyst one sees the delicate anterior wall in the narrow intense beam of the slit lamp, but a hole has raised edges that bulge forward and no anterior wall can be seen. Eyes showing this change frequently have a

surprisingly good degree of visual acuity, and it is quite wrong to treat them as a "prophylactic measure" with light coagulation of the fovea; this operation is not only unnecessary, but may even be harmful. There is nothing that needs to be prevented, and there is a risk of making the vision considerably worse.

One vitreous change is especially prone to cause retinal detachment—*i.e.* loss of vitreous during cataract extraction. In adults, however, this usually occurs only when, after a previous vitreous detachment, the posterior vitreous membrane becomes caught in the wound. This membrane later becomes taut and may cause retinal tears and detachments which are very difficult to treat (Fig. 10*a,b*).

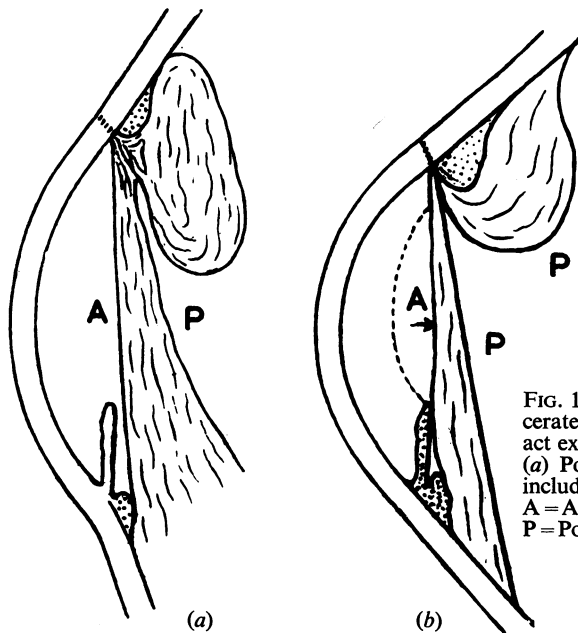


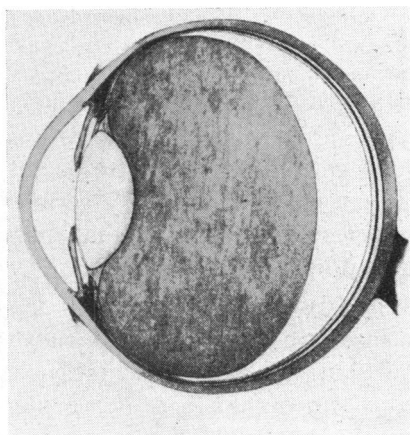
FIG. 10.—Diagram of vitreous incarcerated in operation scar after cataract extraction.  
(a) Posterior vitreous membrane not included in scar (b) included in scar.  
A = Anterior vitreous membrane.  
P = Posterior vitreous membrane.

Besides the form of vitreous detachment which is typical of myopia and old age (*i.e.* vitreous detachment with collapse, Fig. 11, opposite), there is another form which is more frequent in young people (*i.e.* detachment without collapse), but I do not propose to discuss the latter condition in the present paper.

(2) INTRA-OCULAR TUMOURS.—To find no hole in a detached retina without inflammatory signs formerly posed a tricky question in diagnosis, as the condition might be due to idiopathic detachment or to a tumour of the choroid with secondary detachment. The possibility of examining the posterior parts of the eye with the slit lamp makes the differential diagnosis simple. An eye with a retinoschisis should never again be enucleated because of a suspected tumour, for any tumour is clearly visible behind the retinal detachment.



FIG. 11.—Vitreous detachment without collapse (after Hruby).



Yet intra-ocular tumours still offer diagnostic problems. From the standpoint of biomicroscopy, two groups of tumours may be distinguished: those accompanied only by retinal detachment, and those showing further retinal changes (cystic oedema, cloudiness of the retina lying on the tumour, or retinal haemorrhages).

(1) The diagnosis of a malignant melanoma of the choroid is easily made if a compact, opaque tumour with a smooth surface is seen to be situated behind a more or less detached retina. If the tumour is transparent, cystic formations in the choroid must be excluded, but this is a very rare condition.

(2) The second group is not uniform and therefore harder to differentiate. Slow-growing malignant melanomata of the choroid of fairly long standing may either produce haemorrhages or lead to a cystic oedema of the retina, especially if they lie at the posterior pole. Haemangiomas of the choroid, usually situated close to the papilla, soon lead to cystic retinal oedema. It is only by a lucky chance that dilated vessels are seen in such a tumour. In metastatic carcinomata, mostly flat tumours, the retina over the tumour is more or less cloudy, so that the differential diagnosis from chorioretinitis must be considered; but in cases of choroidal metastasis the distinct prominence of the choroid does not correspond with the moderate retinal cloudiness and the absence of inflammatory change in the vitreous in front of the focus.

If the history reveals a mammary carcinoma or a carcinoma of the bronchial tree, diagnosis is easy, but if such indications are lacking, diagnosis may be exceedingly difficult. In one case of this kind, the diagnosis was reached in the following way:

A young man aged 34 years was sent to us with the diagnosis of a tubercular chorioretinitis resistant to treatment. In the macular area a flat speckled prominence of the choroid was seen, and in front of it a cloudy and flat detached retina. There were no inflammatory cells in the vitreous. There was a history of haemoptysis. The suggested diagnosis was a suspected metastasis from a carcinoma, but general examination, especially of the thorax, revealed no signs of a primary tumour. Another possible diagnosis was an early malignant melanoma of the choroid. Since these tumours rarely react to x-ray treatment, whereas metastatic carcinomata of the breast and the bronchial

tree respond well, the eye was treated with 3,000r radiation. Within a short time the tumour disappeared and the visual acuity improved to 1·0. The diagnosis of a metastatic carcinoma from the bronchial tree was thus confirmed. A suspicious spot was then found by bronchoscopy and 6 months later the patient died of carcinoma of the bronchus.

The differential diagnosis between malignant melanoma and haemangioma of the choroid still seems to be difficult and unsatisfactory, above all because we have to be absolutely certain before we exclude the suspicion of the former on account of its malignancy. In this field a great deal still remains to be done.

(3) CHORIORETINITIS.—This condition may be accompanied by cells in the vitreous in front of the focus of inflammation, but every case of choroiditis does not inevitably lead to this cellular exudation. Biomicroscopy shows that there is every possible gradation between a slight inflammatory choroidal oedema not affecting the retina, and intense retinitis with pre-retinal pockets of exudate. The slight cases are the most interesting, though by no means slight from the patient's point of view. It is especially in cases of slowly progressive serpiginous choroiditis at the posterior pole of the eye that the slit lamp reveals that they do not heal completely for months and that thickenings remain at the edges of the foci, even if the process appears to be healed when viewed with the ophthalmoscope. It is from these areas that the disease starts again after an interval of a few weeks; this is not a recurrence, but an advance after a temporary stationary phase (Fig. 12). An apparently healed focus of juxtapapillary chorioretinitis may show a precipitate-like granuloma (Fig. 12*b,c*); this may remain quiescent for months or even years, and may then break out again and lead to subsidiary foci. A great many cases of so-called "relapsed" choroiditis, and perhaps of anterior uveitis also, do not represent a fresh dissemination of antigen attaching itself again and again at a chosen site, but are reactivations of latent unhealed foci of inflammation.

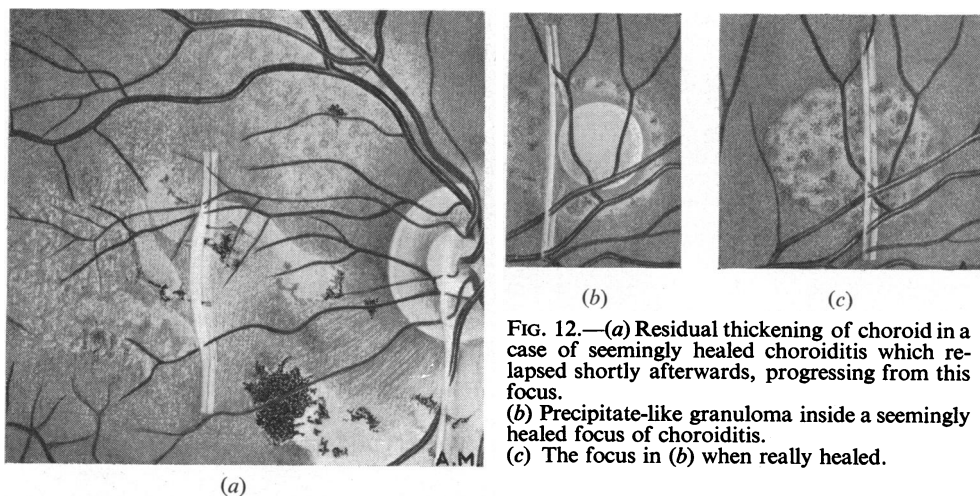


FIG. 12.—(a) Residual thickening of choroid in a case of seemingly healed choroiditis which relapsed shortly afterwards, progressing from this focus. (b) Precipitate-like granuloma inside a seemingly healed focus of choroiditis. (c) The focus in (b) when really healed.

Fig. 13 (*a,b*) shows that biomicroscopy may show up foci that cannot be seen with the ophthalmoscope. In both eyes in this patient there were curious cystic formations in the retinal pigment-epithelium which had resulted in serous retinitis. Nothing but a retinal oedema could be detected with the ophthalmoscope, but with the slit lamp the retro-retinal cysts immediately became visible to the observer. In this case the condition was probably due to toxoplasmosis.

FIG. 13.—Retro-retinal cystic formation covered by an oedematous retina which could be seen only with the slit lamp. (*a*) Retinography.

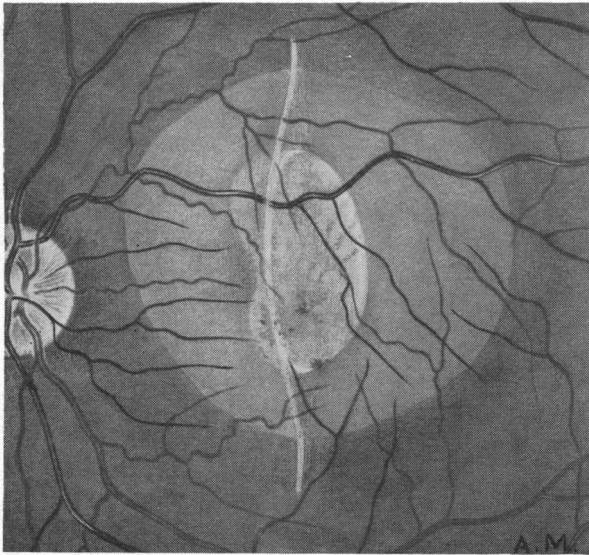
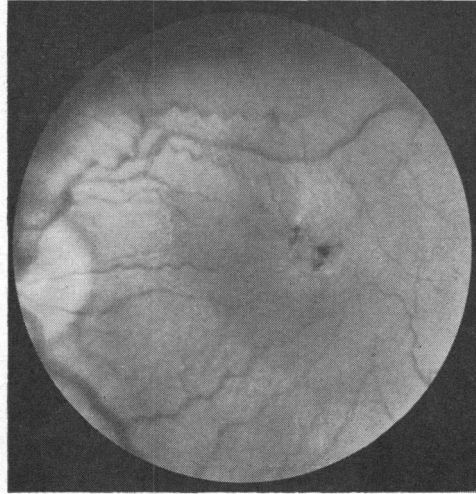


FIG. 13(*b*) Slit-lamp picture.

(4) PAPPILARY CHANGES.—One of the most important problems of glaucoma is the mechanism of glaucomatous excavation. Does the raised intra-ocular pressure push the lamina cribrosa out backwards, and do the

nerve fibres degenerate because they are thereby stretched across the sharp rim of the scleral canal? Or is nerve fibre degeneration with formation of Schnabel's caverns the first stage, and does the bulging outwards of the lamina cribrosa follow as a secondary phenomenon?

The latter explanation would mean that the glaucomatous excavation is only an indirect result of the raised pressure, but the former would mean that the fibrillar degeneration is an immediate result of the raised pressure. Our histological material is extensive, but seldom comes from eyes that were not already blind as a result of absolute glaucoma, so that the findings give little help in solving the problem of early papillary changes.

Biomicroscopy enables the distance between the lamina cribrosa and the retinal level at the edge of the papilla to be measured in normal eyes and also in glaucomatous eyes with typical excavation which still retain a fair degree of visual function. These measurements are the same in both groups and this indicates that the glaucomatous excavation is not due to pressure on the lamina cribrosa. The condition must therefore be caused by atrophy of the papillary nerve elements including the glia, which lays bare the scleral canal inside the lamina cribrosa. Where there is no well-developed scleral canal, no glaucomatous excavation can occur, as in cases of high myopia with extensive temporal or circular conus. In such cases simple atrophy develops instead of the glaucomatous excavation, and because the Schiötz tonometer shows low pressure values with bizarre visual field defects due to myopic fundus changes while glaucomatous excavation is lacking, the presence of simple glaucoma goes unrecognized.

It is usually stated that simple glaucoma is very rare in high myopes and at the same time it is accepted that such patients not infrequently go blind through "myopic degeneration" of the retina and optic nerve. This is not correct; the majority of those myopic eyes which become blind in old age do so because of unrecognized simple glaucoma. Our investigations seem to show that simple glaucoma is especially frequent in high myopes, but only systematic applanation tonometry will reveal the true disease.

It has been my aim in this lecture to indicate how a number of problems in clinical ophthalmology may be elucidated by biomicroscopy of the posterior parts of the eye. This field of investigation has not yet been completely surveyed and there are still many possibilities for further study.